Giant tuberculoma mimicking malignancy: A Case Report

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Abstract - Tuberculosis continues to be a significant health concern globally, particularly in regions with limited healthcare resources. While pulmonary tuberculosis is the most common form, extrapulmonary manifestations, including tuberculomas, present unique diagnostic challenges, especially when mimicking malignancies. Here, we report a rare case of a giant tuberculoma mimicking malignancy in a 7-year-old boy. The patient presented with nonspecific symptoms, including fever, weight loss, and headaches. Imaging studies revealed a large intracranial mass lesion suggestive of malignancy. However, histopathological examination following surgical excision revealed features consistent with tuberculoma. The patient responded well to anti-tubercular therapy and showed significant clinical improvement. This case underscores the importance of considering tuberculosis in the differential diagnosis of intracranial mass lesions, particularly in endemic regions, even in paediatric patients. Early recognition and appropriate management are crucial for favourable outcomes in such cases.

Keywords: Tuberculoma, tuberculosis, paediatric, intracranial mass, differential diagnosis, case report.

INTRODUCTION
Tuberculosis (TB) remains a major global health concern, affecting millions of individuals each year, particularly in resource-limited settings [1]. While pulmonary TB is the most common presentation, extrapulmonary manifestations, including tuberculomas, pose unique diagnostic and therapeutic challenges, especially when they mimic malignancies. Tuberculomas are granulomatous lesions that can affect various organs, including the brain, and their presentation may vary widely, making diagnosis challenging, particularly in paediatric patients. Intracranial tuberculomas, although relatively rare, can occur in children and are associated with significant morbidity and mortality if not promptly diagnosed and treated [2,3]. Clinical manifestations of intracranial tuberculomas often include nonspecific symptoms such as headache, fever, seizures, and focal neurological deficits, which can easily be misinterpreted as signs of other intracranial pathologies, including neoplasms.

CASE REPORT
A 5 year old male child presented to the emergency department with generalised tonic clonic seizures, which was managed with the course of antiepileptics and oxygen therapy. After the seizures subsided, patient difficulty in opening his eyes and groaning in pain and his vision was blurry. Patient was then shifted to neurosurgery department with history of complain of headache, multiple episodes of vomiting along with on and off high fever for the past 8 days. No history of past of trauma or chronic medical illness. History of tuberculosis in the family was taken to which the father denied any history of TB contact. Patient on examination was underweight for age, very thin built and lethargic. The child had lack of appetite and did not speak and expressed himself by unsensible sounds.

The patient was sent for radiological examination. MRI brain revealed focal T2 hypointense lesion in superior part of right cerebellar hemisphere measuring approximately 29 X 21 mm with mild locoregional oedema, causing severe pressure effect over adjacent part of fourth ventricle with mild supratentorial ventriculomegaly. No diffusion restriction seen. No obvious blooming seen. Imaging features are suggestive of Inflammatory aetiology (? Giant Tuberculoma) [4,5]. Less likely imaging measuring possibility may also include Lhermitte duclos disease.

Patient was operated in the neurosurgery department for hydrocephalous shunt surgery three times to decrease the pressure effects. Biopsy specimen was sent to Department of pathology for histopathological analysis. Gross specimen received were multiple irregular tissue bits, greyish white to greyish brown in colour, soft to firm, altogether measuring 4 X 5 X 2 cm. Microscopic findings revealed histological section showing sheets of marked caseous necrosis with epitheliod granulomas and multiple langhans giant cells with chronic inflammatory infiltrate with congested blood vessels. Histological finding are consistent with Tuberculoma. CSF aspirated during surgery was sent for CBNAAT examination, and was detected to positive. Patient was started on anti-tubercular treatment (ATT) and is responding well to it and recovering.
Figure 1: Tuberculoma A-C: (A) Gross image showing multiple bits of excised mass (B) Low power magnification (10X, H&E slide) histopathology slide showing epithelioid cells, macrophages, and chronic inflammatory infiltrate with areas of necrosis. (C) High magnification (40X, H&E slide) histopathology slide showing epithelioid cell with chronic inflammatory infiltrate

DISCUSSION
Cerebral tuberculoma is a dreaded presentation of tuberculosis that results from hematogenous spread of a distant tuberculous focus. Cerebral tuberculomas may be solitary or multiple and are most commonly seen in the basal parts of the brain. The typical gross features of tuberculomas are small, round, or oval-shaped nodules, ranging from 2 to 12 mm in size. There are only a few published case reports describing giant cerebral tuberculomas in the literature. It is extremely rare to find cerebral tuberculomas that are large enough to produce compressive features. Giant cerebral tuberculomas can be easily misdiagnosed as intracranial tumors. Only 30% of patients with cerebral tuberculomas have a suggestive chest radiograph. Furthermore, a cerebrospinal fluid (CSF) analysis may not be contributory, as tuberculosis bacilli are not always observable in the CSF. In our case, we could establish the diagnosis of tuberculoma only after a histopathological examination of the excised lesion.

The neuroimaging picture for cerebral tuberculoma is different according to the type of granulomatous lesion. If the tuberculoma comprises granulomas that are non-caseating, the lesion is usually hypointense or isointense to gray matter on T1-weighted images, and hyperintense on T2-weighted MR-images. Caseating granulomas typically have a solid centre that is hypointense or isointense on T1-weighted images and isointense on T2-weighted imaging. A central region of T2-hypointensity can be seen because of gliosis and monocyte infiltration. This is a useful finding, as we do not find it in many other intracranial lesions. In post-contrast imaging of tuberculoma, we typically see a peripheral ring-enhancement because of vasogenic edema. Sometimes, central liquefactive necrosis can occur in tuberculoma, which can make differentiation from a cerebral abscess difficult. Some reports have described the ‘target sign,’ which is a ring-enhancing lesion with an additional central area of enhancement or calcification, as characteristic of cerebral tuberculomas. However, this is a nonspecific finding related to central enhancement and may lead to an erroneous diagnosis of cerebral tuberculoma. If available, MR spectroscopy is useful to differentiate tuberculomas from other lesions. In tuberculomas, typical MR spectroscopy findings include a decrease in N-acetylaspartate/creatine and prominent peaks of lipid and lactate. Cerebral tuberculomas should always be considered in the differential diagnosis of solitary intracranial mass lesions.

However, the diagnosis is difficult because the neuro-imaging presentation is varied and can be non-specific. In many cases, a definitive diagnosis can only be established with a biopsy of the CNS lesion for histopathology and acid-fast bacilli stain and culture. Antitubercular drugs are usually effective in treating cerebral tuberculomas. A long course of chemotherapy for nine to 18 months is typically required, but total cure rates are high. Surgical intervention may be necessary for situations with acute complications such as obstructive hydrocephalus, large lesions with significant mass effect, brainstem compression, or when the diagnosis is not ensured.

CONCLUSION
In regions where tuberculosis is endemic or in patients with a history of prior tuberculosis exposure or infection, cerebral tuberculomas should always be considered in the differential diagnosis of single and multiple space-occupying brain lesions. However, because of its rarity, a giant cerebral tuberculoma can easily be misdiagnosed as an intracranial tumor such as a high-grade glioma. A timely and accurate diagnosis allows the early administration of anti-tubercular drugs, decreases patient morbidity, and can potentially prevent neurosurgical excisions that are not required in all patients.

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REFERENCES: